not continue long enough in that locality. From this time on the spread of the blebs was rapid, until finally the whole body was covered, some of the blebs being two to three inches long. Even the vulva and anus were implicated, the lesions in this region giving rise to a great deal of pain. Drs. Regensberger and Montgomery saw the patient, and confirmed the diagnosis. Numerous measures were resorted to to relieve the patient's distress. Among other things she was wrapped in blankets which had been dipped in oil, but the annoyance of these was so great that she was returned to her ordinary nightclothes, and all the surfaces liberally covered with dusting powder. At last opiates were resorted to.

In view of the patient's age, of the fact that the blebs quickly became saggy and filled with pus, and, most of all, because the process had begun on the mucous surfaces, the prognosis was made infausta. This was confirmed by the patient's death taking place on Dec. 8th, about ten weeks after the first lesion had appeared. The extreme annoyance of having such an extensive area involved together with the impossibility of conveying nourishment to the patient (even nutrient enemata not being tolerated) were, without doubt the cause of the fatal ending.

This was the only case of pemphigus affecting the mucous surfaces that I had ever seen, and I may never see another, so rare are the cases. It is worthy of notice, in this connection, that my colleague, Dr. R. D. Cohn, has a parallel case to report to you at this meeting.

Having lost all my notes, I can not give exact data on this case. I know there was some fever, but I remember that it never exceeded 103.

I have seen several articles of late bearing upon the diagnosis of dermatoses when occuring on the mucous surfaces. A late number of the J. A. M. A. contains a very good article by Dr. Linn Emerson, of Orange, N. J., on the appearance of lichen planus in the mouth. If these lesions are confined to the mucous surfaces, and remain confined to that locality for years, as they do in some cases, they almost defy diagnosis. Owing to the thinness of the covering and the moisture to which they are constantly exposed the lesions present hardly anything characteristic. Moreover, their rarity makes familiarity with them an impossibility.

MYOCARDITIS: ITS PHYSIOLOGY, PATHOLOGY, SYMPTOMS AND TREATMENT.

By NEIL DONALD GUNN, M. D. C. M., Pacific Grove.

In taking up the subject of myocarditis, I do so with a certain amount of apology, as the specialist may feel that it is a well-trodden path, but the general practitioner is, after all, the final court of appeal, and to such an one this paper is especially addressed. It is a subject that embraces nearly all heart symptomatology, and when we speak of such conditions as dilatation, hypertrophy and high tension, we are but dealing with entities or signs of a general vascular condition, that condition being usually summed up in the general term myocarditis. This term is more or less a misnomer, for it not only includes inflammatory conditions but also degenerations; the fact is, the latter embrace by far the greater number of pathological changes found. When, after many years of doubt, the clinician had evolved a working hypothesis to explain these various heart phenomena, physiology departed from its beaten paths and began to blaze new trails, and in the enthusiasm born of youth and inexperience, promised to clear up all that was obscure. Time has proven how much these newer methods have yet to develop before we can with confidence assure ourselves of what is taking place in the circulatory system.

Experimental physiology began most naturally on the circulation and the various physical and mechanical forces employed in propelling blood. The various reasons why the heart beats, offered since the discovery of the circulation, would fill a paper of some dimensions, and if one were to follow the arguments in favor of each explanation, it would occupy a volume. With all due respect to the dead and the living, "that vast army of experimentalists," we are still looking for light and still presenting problems that are unanswerable. Are we any nearer the cause of the heart beat and its various disturbances that were Bright and Brestowe? It is of interest to follow the various and varying moods and tenses of this question, and only a few references can here be made to the physiological work that has been done.

When Remak in 1844 discovered the ganglion in the heart walls, he founded a school that claimed that the heart beat was due entirely to nervous influence; Ludwig in 1848 described another group of nerve cells; then Bidder demonstrated yet another. As methods of research improved, Dogiel and Gerlach showed that ganglionic cells could be found in nearly all parts of the heart; Freidlander and Schweiger-Seidl and Valkman arrayed themselves with this school. These were the founders of a school that still has many advocates.

This theory seemed to satisfy clinician, physiologist and anatomist. The view certainly seems rational, as the great number of nerve centers and complex sympathetic network must necessarily have some function. By stimulating the various nerves connected with the heart the number of beats could be reduced or increased almost at will. Trophic disturbances could also be produced in the heart muscle, and what more was there to solve?

Engleman, an acute observer, happened while experimenting on an animal to notice that there was a rhythmic contraction of the ureter when all the nerves were severed; a segment of the same ureter would continue this rhythm; this led to the founding of a new theory, viz., the innate contractile nature of muscle.

Gaskell in England immediately began to study the muscle of the heart and showed that the vagus was not, at least constantly, an inhibitor of the heart, and when his great disciple, Martin, kept a mammalian heart beating outside the body for some months the myogenists seemed to have the best of the argument. This latter theory has been greatly strengthened by Professor Loeb of Berkeley,.

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whose work on the influence of certain salts on the contraction of muscle has opened a wide field both to the physiologist and the clinician; and, though these chemic stimuli have not yet put us in possession of facts to warrant our feeding the heart certain ions, yet I see no reason why such treatment may not be later adopted.

If I might say a word "en passant" to those who are interested in heart work, it would be to study more closely the relations of the heart to the fluid it contains. An old theory and one I fear yet insufficiently abandoned "is that the heart is the last to suffer in wasting diseases," when as a matter of fact it is the first to suffer. Those who are interested in the myogenic theory would do well to consult the work of Cyon, who is the ablest exponent of this theory.

There is a late development in the anatomy and physiology of the heart which I must mention and one which I fear has passed like a brainstorm over our profession. This theory gives to the heart a sort of brain or a central distributing and conducting organ known as the "bundle of His." Many clinicians and not a few of the physiologists would have us believe that here lies the center for the control of blood tension heart rhythm; in fact, the sum total of the heart's activities. The probabilities are that there is more or less truth in all these contentions, but not all the truth in any one.

Comparative anatomy would lead us to the conclusion that in the lowly organized heart the beat is entirely due to the contractility of the muscle; but in the highly organized heart, as found in all mammals, it becomes necessary to have a nervous control. That is, conditions of temperature, digestion and muscular action are so complex and the calls made upon the heart so varied that there must be some unusual means of increasing or slowing the beat, and also of raising or lowering blood tension. This must necessarily be done by some mechanism connected with but not necessarily a part of the heart muscle.

In referring, again, to the "bundle of His" I may say that it will in no way conflict with the theory just advocated, but rather supports it. This bundle lying in the auriculo-ventricular walls is certainly a distinct anatomical body having its own capsule, surrounded by its own ganglia and closely related to the endocardium; paler in color than the adjoining muscle, connected with each heart cavity, it certainly must be thought of both in physiological and pathological conditions. When the final word is said, however, I think this muscular and nervous bundle will have shrunken in significance relative to its anatomical proportions. I will have more to say concerning this bundle when speaking of the pathology of the heart.

Pathology: Virchow was the first to describe acute parenchymatous myocarditis, a condition which he commonly found associated with all fevers. Zenker, who did much work on this acute condition, held that the change was due to the high temperature, and the higher the temperature the more liable was the heart to suffer. This view was at once adopted by the practitioner, and efforts were made to keep the temperature down by all available means. The manufacturer of antipyretics reaped a harvest and the undertaker was made rich by the depressing effects produced on these weakened hearts; Osler was the first, I think, to suggest that the toxines and not the temperature caused the degeneration; it was Renault who proved this condition to be a parenchymatous change rather than inflammatory, as Virchow stated. This change was always accompanied by a segmentation and fragmentation of the fibre, also an enlargement and fragmentation of the nucleus, whereas the cement substance is somewhat dulled in appearance, more or less granular looking and swollen.

The heart on inspection is changed in color, size, and consistence, being described by some one as a sort of "dead leaf" color. Hektoen insists that it is neither the heat nor the toxines, but a want of proportion between the amount of work required and the ability of the fibres to do this work. In other words, a worn-out condition of the fibre; a good reason, by the way, why we should lessen the work put on the heart. It necessarily follows that if overwork can produce this change in a weakened fibre a great deal of overwork would produce the same change in a healthy fibre, and such we find to be the case in heart strain.

Daland in summing up recent literature on myocarditis quotes Fedeshi as having produced this condition by cauterizing a healthy heart, and, killing the animal a few days later, found conditions exactly resembling those present in the acute infections. This would lead us to the conclusion that any injury to the heart muscle may result in parenchymatous change. Just how far this degeneration can go without inducing permanent damage cannot at present be made out, but we are realizing more and more that many hearts which were never suspected and many more than were considered cured deceived us and the regeneration was not complete. In fact, many claim that this change is never wholly recovered, but leads to a progressive degeneration. This, I take it, is an extreme view.

The latest pathological announcement relates to the muscle "bundle of His," which I mentioned previously. The claim is made that in acute infectious diseases there is not a general myocardial change, but in a great number of cases, at least, the change is confined to this conducting bundle. They do admit that diphtheria does produce a general and acute rheumatism, a localized spotted subendocardial degeneration, but not so the other acute infections, and in 112 cases of acute heart failure examined by Tawara it was this bundle that was at fault in every case.

Erlangen of Johns Hopkins in his experiments proves that pressure on this bundle may produce irregularity, intermittency and even death; so that by analogy we may reason that an inflammatory deposit or vascular change in this bundle might do much damage. If these observations and experiments be true, then we must forsake, at least in part, our old position; but that the heart walls and the

valves take some part in these pathological changes seems to me undoubted.

Chronic myocarditis is comparable to arteriosclerosis, some fibre bundles being much more affected than others; the tendinæ and papillary muscles suffer most, especially those in the left ventricle. The ventricular wall subjected to the greatest strain usually suffers most, but other questions of interference with the circulation, etc., have to be considered; that overwork and high tension are responsible for many of these changes is as true as in arteriosclerosis.

The coronary arteries decide in great masure the condition of the heart walls, as they preside over nutrition. If one coronary is more affected than another, the corresponding side of the heart suffers most. The left coronary, owing to its situation, is most frequently affected, and for this reason left-sided heart trouble is more common than right.

This paper will not admit of the discussion of brown atrophy and amyloid heart or the granulomata.

Symptomatology: Louis of the Charite was the first to describe the sequence of physical signs in the acute infectious heart. The first sign is accentuation of the pulmonary second sound. This is explained by overfilling of the auricle, due to a weakened ventricle. Second, mitral insufficiency, due to dilitation, accompanied sooner or later by a mitral murmur at the apex. Third, reduplication, due to increased pulmonary and lessened systemic tension, causing the aortic to close later than the pulmonary valves. Fourth, when the intoxication is uncontrolled and the disease runs into weeks, there comes a systolic basic murmur called by Potain cardiopulmonic murmur. This murmur has stirred a good deal of discussion as to its causation, and is usually considered due to dilitation of the ventricle and auricle. A similar murmur is sometimes found in apparently healthy hearts, but you can make it disappear by asking the patient to stop breathing. It is not always found even in advanced cases, and it seems to me that a peculiar sagging and a change of relations between the heart and the great vessels is responsible for this bruit. The pulse rate is by no means constant, sometimes slow, sometimes fast, and occasionally irregular, the slightest movement often causing great acceleration of the pulse or even a sudden asystole. This is especially noticeable after pneumonia and diphtheria. Irregularity of pulse in these cases is not common and heart block, which has become so fashionable recently, has been in my experience a rare symptom.

Percussion often reveals little, as the dilatation is not sufficiently pronounced to be made out, but with much care the right heart will be found more or less dilated, the left ventricle being the last to enlarge.

Chronic myocarditis may be of many years' standing before discovery, and sudden failure leading to death may be the first suspicion that we have a heart lesion. These cases usually, however, begin their symptoms by palpitation, shortness of breath, indigestion and general muscular weakness.

Tachycardia, or the runaway pulse, may be the first evidence of a diseased condition. Such cases, as a rule, have a serious prognosis, as the great amount of overwork due to the rapid pulse dilates the heart beyond possible recovery, and palliation is all that we may hope for. Bradycardia, or very slow heart, is in my experience a somewhat rare beginning and is not so grave as a very fast heart. Arrythmia, which we sometimes find as a functional disorder, usually accompanies dilatation, especially of the auricles; whether the "bundle of His" really controls the rhythm of the heart, still needs confirmation.

Asthmatic attacks, sometimes called heart asthma, is a fairly common symptom, and such a symptom, appearing late in life, should always lead one to examination of the heart. Digestive disturbances due to engorgement of the portal vessels are quite common. Albuminuria is quite a common condition, and it is often hard to decide whether due to renal engorgement or to an actual renal disease.

Changed mental conditions and delirium are usually late and dangerous manifestations. Cheyne-Stokes respirations, a late manifestation, are as a rule, but not necessarily immediately fatal. Oedema and general anasarca usually come sooner or later. If the right heart be affected, then the pulmonary symptoms predominate—dyspnoea, rales, cough dilated jugulars, with venous pulse in the neck, etc.

On examination of the heart one finds enlargement and dilatation of varying degrees, with dilated valves and regurgitant murmurs. One must not forget that if the heart musculature become extremely weak, murmurs may disappear and return again as the muscle tone increases. The right heart may remain fairly good for a long time after systemic symptoms show themselves and vice versa. The intensity of the murmur offers no reliability as to the gross lesion. In those large hearts accompanied by contracted kidney there is still discussion as to the real cause of the trouble. Bright was of the opinion that the increased blood tension and hypertrophy were due to circulating poisons. Traube, on the other hand, put it down as a simple physical problem due to greater force being required to force the blood through the kidney.

It is a question whether simple parenchymatous changes in the kidney do produce a rise of blood pressure. The toxic theory has of late revived and diseased glomeruli are said to be responsible for the improper filtering of the poisons circulating in the blood; these poisons being responsible for the heightened blood tension.

There are cases of arterio-sclerosis and enlarged heart where the kidney seems perfectly healthy, but that there is a renal inadequacy in these cases is undoubted, as the blood pressure can be immediately lowered by lessening the nitrogenous diet or by a purely milk diet.

Treatment.

The acute infectious heart needs the greatest care; elimination of the poisons by keeping skin, kidneys and bowels active; absolute quiet in a recum-

bent position; as little moving in the bed as possible; no talking and no excitement; one day and one night nurse and no one else allowed in the room.

Antitoxine in diphtheria and streptococcic infections is of great value. Digitalis, if at all, should be given with caution. Spartein acts well and does not produce restlessness as strychnin sometimes does. Strychnin must always be considered, and is, one of our sheet anchors. Nitroglycerin in a crisis, but its action is so brief that its continued use is not usual. Alcohol may sometimes do good, but as a rule is harmful, and should be given with the greatest caution. Ammonia and its various salts are good stimulants and are followed by no bad effects unless it be on the stomach. After the danger point has passed, diet must be free, iron and bitter tonics administered and the convalescence prolonged for some weeks after all signs have disappeared.

In chronic cases, to use an Irish bull, the best treatment is to prevent the disease. These cases often have a long premonitory period, shown by high tension, pulse plethora and a tendency to a fat accumulation. It is here that a bit of good advice may save trouble. Smoking, drinking and overfeeding, especially of nitrogenous food, should be strongly condemned. High tension pulse is usually possible of diagnosis by the trained finger, but the sphygmo-manometer is much safer and with a little experience gives fairly accurate information.

Even after beginning degeneration in the heart and blood vessels, much good may be accomplished by regulation of the diet, changing of habits, the administration of iodides and nitrates and by nauheim baths. Cold bathing, in these cases, should be avoided; this is especially true of the fatty heart. Exercising of a quiet and unexciting kind needs to be recommended, such as golf playing and slow walking on the level. Hill climbing as recommended by Oertel, should be under the close supervision of a physician.

When the breakdown comes and compensation fails, then absolute rest in a recumbent position between blankets, in a temperature kept as near uniform as possible, must be enjoined. The food must be simple and easily digested, and if the kidneys are affected, the plain milk diet. Schott baths are of great value in slowing and regulating the pulse and dilating the superficial vessels. The resistance movements which go with these baths are of benefit, but should be given by a trained assistant.

Drugs.—Digitalis stands pre-eminent, but it must be of proper strength and proper preparation. The usual tinctures and tablets vary greatly in strength and composition, and many failures are due to an ignorance of this fact. The alkaloids of digitalis are not comparable to the whole drug. The English leaves should always be used, as they are stronger and much more uniform in composition than any other variety. I usually give infusion made fresh each time. Of the powder, one to three grains given in each dose. Boiling water is poured on the leaf or powder, allowed to stand from 20 minutes to half an hour, pour off the infusion, add cream and sugar, or lemon to taste and drink as ordinary tea three to four times a day after food. When given in this way it seldom disagrees with the stomach and scarcely ever fails to produce results. It is a double-edged sword, however, and must be watched. The patient should not be going about his daily duty when taking it. If the pulse becomes too slow or after being slow suddenly gets fast or if urine begins to diminish, then stop the drug for a few days. Spartein is of great service in doses of ¼ to ½ grain, alternating with strychnin every three or four hours. Strophanthus and the many other heart remedies may be tried, the former may sometimes add to the efficacy of digitalis. In full-blooded cases active purgation and stimulation of the kidneys are of assistance. Diuretin is especially useful in relieving dropsy and edema when the kidneys are healthy.

In greatly dilated hearts with very irregular pulse, general edema, etc., there is nothing that gives the relief which free bleeding does, and this may be repeated as occasion demands, being careful not to produce acute anaemia. Where there is abdominal dropsy, resort to tapping and repeat at intervals, being careful not to too suddenly lessen intra abdominal pressure.

Morphin, though a heart tonic of great value, must be given guardedly, especially where the kidneys are at fault. Where there is great restlessness and delirium nothing acts so well as morphin in ⅛ to ¼ grain doses.

The nutrition must be watched, especially in cases that have been depleted for a long time. We should give plenty of wholesome, easily-digested foods, with the addition of dry wines or a little good whisky. If the patient improves, massage and carefully-supervised resistance movements should be given. Business affairs all settled, there may be a long period of comfortable invalidism.

INJURIES OF UPPER END OF FEMUR IN ADOLESCENTS AND CHILDREN.*

By JAMES T. WATKINS, M. D., San Francisco.

On April 17, 1905, I saw J. W. of Eureka at my office. He was 13½ years old, moderately tall and weighed 152 pounds. He had never had any severe illness; but six weeks earlier a wagon had been backed up against his left hip, causing him to be thrown back against the side of the barn. He did not fall down, and although he complained of pain in his thigh and knee, neither then nor later did he go to bed. While in bed or lying down, he was free from pain. Still gradually his limp and pain had increased up to the date of his coming to see me.

Inspection showed a very much too heavy adolescent. He lay with his limbs in full extension, and with a very little more outward rotation on the affected side. Still the contours of the two sides were practically symmetrical. Measurements showed an atrophy of about a centimeter and a half of the affected thigh. There was about the same amount of shortening. The trochanter appeared to be a little above Nelaton's line, but the boy was so fat that this point was hard to determine. There was a little limitation of motion in all directions, most

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